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THE IMPACT OF TRACHEOTOMY ON THE CLINICAL COURSE OF VENTILATOR-ASSOCIATED PNEUMONIA

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SUMMARY – Ventilator-associated pneumonia (VAP) is the most common infection among intensive care unit (ICU) patients. The aim of the present study was to evaluate the impact of tracheotomy on VAP clinical course. The study was conducted in a 15-bed Surgical and Neurosurgical ICU, Department of Anesthesiology and Intensive Care, Sestre milosrdnice University Hospital Center in Zagreb, Croatia. All patients developing VAP during ICU stay were eligible for the study. In VAP patients not tracheotomized during ICU stay, the mortality rate was approximately two times higher as compared with patients tracheotomized either before or after VAP onset (crude risk ratio 1.83, 95% confidence interval (95% CI) 1.15-2.91, p=0.01; crude odds ratio 3.47, 95% CI 1.52-7.94; p=0.003). In the surviving VAP patients, the duration of mechanical ventilation before VAP onset was higher in the "T before VAP" group as compared with the "no T before VAP" group (8, 6-10 *vs.* 3, 2-5; p<0.001), but the number of post-VAP days on mechanical ventilation was shorter in "T before VAP" patients than in "no T before VAP" patients (0, 0-1 *vs.* 4, 3-9; p<0.001). The duration of mechanical ventilation after VAP onset in the "T after VAP" group was longer as compared with the "T before VAP" group (4, 3-12 *vs.* 0, 0-1; p<0.001). The present study indicated tracheotomy to be associated with a reduced duration of mechanical ventilation after VAP onset, but only if patients were tracheotomized at the moment of VAP onset.

Key words: *Critical care; Tracheotomy; Pneumonia, ventilator-associated; Prognosis; Survival analysis; Treatment outcome*

Introduction

Ventilator-associated pneumonia (VAP) is the most common infection among intensive care unit (ICU) patients[1,2]. VAP is defined as a type of nosocomial pneumonia occurring more than 48 hours after intubation and mechanical ventilation. VAP occurs in 8%-28% of all intubated patients[1]. Generally, surgical ICUs have higher VAP rates as compared with non-surgical ones[3]. VAP is the leading cause of ICU mortality, with the reported mortality rates ranging from 24% to 76%[1]. Microorganisms most commonly responsible for VAP onset are *Pseudomonas aeruginosa*, *Staphylococcus aureus* and *Enterobacteriaceae*, but causative agents widely differ depending on the ICU patient population, duration of ICU stay, VAP onset point, underlying diseases and hospital settings[1]. A number of risk factors may favor VAP onset, the most commonly reported among them being duration of mechanical ventilation (MV), enteral feeding, number of re-intubations, severity of illness (Acute Physiology and Chronic Health Evaluation II score, APACHE II score), underlying pulmonary disease, supine body position, depressed consciousness, prior exposure to antibiotic therapy, tracheotomy, etc.[2]. The role of tracheotomy in VAP development remains controversial. While tracheotomy may protect against

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VAP because it facilitates bronchial toilette, reduces longstanding epithelial injury and improves patient mobility as compared with endotracheal intubation[4], it may, at the same time, increase the risk of VAP onset because of the direct injury to the airways and possible insertion of bacteria during the procedure[5]. The impact of tracheotomy on the development of VAP is not clearly defined. Some studies found decreases in VAP rates in patients having tracheotomy[6-12], others have reported tracheotomy to be a risk factor favoring VAP onset[13-20], while some could not demonstrate any VAP-related impact whatsoever[21-24]. Because of the conflicting literature data, there is currently no consensus on tracheotomy as either a VAP-protective or VAP-causative factor[5]. The article by Vello *et al.*[5], which reviews the literature that explores the relationship between tracheotomy and VAP, claims that explicit data on tracheotomy-VAP timeline can be found in not more than 4 studies[8,13,24,25].

The aim of the present study was to evaluate the impact of tracheotomy on the clinical course of VAP, as well as to assess the risk factors associated with lethal VAP outcomes.

Patients and Methods

The study was conducted in a 15-bed Surgical and Neurosurgical ICU, Department of Anesthesiology and Intensive Care, Sestre milosrdnice University Hospital Center, Zagreb, Croatia. The study was approved by the Hospital's Board of Ethics (E.P. number: 35-1/09). Retrospective data were collected from September 2009 to March 2013. Because of the retrospective and observational nature of the study, an informed consent was unnecessary.

All ICU patients that developed VAP during the study period were eligible for the study. Patients were divided into two groups based on VAP onset in relation to tracheotomy time-point ("no T before VAP" and "T before VAP"). The study arm "not tracheotomized before VAP" was divided into "never tracheotomized" ("no T after VAP") and "tracheotomized after VAP onset" ("T after VAP") sub-arms.

Reasons for ICU admission were medical, trauma without surgery or surgery. As for clinical diagnosis, VAP was established on the Clinical Pulmonary Infection Score (CPIS) based on six clinical assessments, each worth zero to two points[26]. A score of more than six was considered suggestive of VAP. The CPIS score was calculated when there was clinical suspicion of VAP (presence of new or progressive infiltration on chest radiography and presence of at least two of the following criteria: fever, leukocytosis and purulent tracheal secretion). Only the first VAP episode was evaluated. Quantitative culture of endotracheal aspirate (ETA) was performed so as to identify the VAP pathogens. A pathogen isolated at a concentration of more than 10^5 CFU/mL was considered causative of VAP. Endotracheal aspirate sample with more than 10 squamous epithelial cells *per* visual field represents an invalid sample[27]. Purulent sputum is defined as secretions from the lungs that contain more than 25 neutrophils *per* visual field.

Patients were tracheotomized surgically or percutaneously. Surgical tracheotomies were performed in the operating room by otolaryngologists. Percutaneous tracheotomies were performed at patient bedside by ICU physicians using the Griggs method.

The exclusion criteria were pneumonia prior to MV or within 48 hours following MV initiation, tracheotomy performed before ICU admission, emergency tracheotomy, and age below 18.

All patients were fed enterally using a nasogastric tube (in most patients starting from the second day of ICU stay), received systemic stress ulcer prophylaxis (ranitidine or proton pump inhibitors) and were kept in a semi-recumbent position during their ICU stay.

The inter-group comparison was based on the patient status at ICU admission and during ICU stay, characteristics of VAP and clinical outcomes.

Statistical analysis

Since continuous variables were not distributed normally, the differences between the groups were analyzed using the Kruskal-Wallis ANOVA followed by the *post hoc* Mann-Whitney U-test (with a modified Bonferroni correction) whenever the ANOVA yielded a statistically significant result. As for categorical variables, the inter-group differences were analyzed using the Pearson's χ^2 or the Fisher test (with Bonferroni-Sidak correction) whenever the overall results of the above tests were statistically significant.

The risk of mortality was analyzed as crude risk and odds ratio, and in the multiple linear logistic re-

gression models, controlled for potentially relevant confounders, i.e. APACHE II score at admission, Simplified Acute Physiology Score II (SAPS II) at admission, tracheotomy, total duration of MV, comorbidities (malignant disease, chronic cardiac disease, and diabetes mellitus), type of surgical patients and use of corticosteroids. In order to maintain the recommended ratio of the number of predictors *vs.* number of study subjects[28], as predictors in the models were chosen variables found to statistically significantly differ between tracheotomized and non-tracheotomized patients (SAPS II at admission, APACHE II score at admission, diabetes mellitus, type of surgical patients) or to be significantly related to studied outcome on univariate analyses (tracheotomy, total duration of MV, malignant disease, chronic cardiac disease and use of corticosteroids), while not being inter-correlated with other predictors.

Statistical analysis was performed using Stata/SE 11.2 for Windows (StataCorp LP, USA), at the 0.05 level of significance.

Results

During the study period, 5071 adult patients were admitted to our ICU. Four hundred and fifty-three (8.9%) of these patients were intubated and mechanically ventilated for more than 48 hours, 178 (39%) of these tracheotomized during their ICU stay. VAP developed in 113 (25%) patients, 98 of these not tracheotomized before VAP onset ("no T before VAP") and 15 tracheotomized before VAP onset ("T before VAP").

Table 1. Characteristics of VAP patients according to tracheotomy (T) status at ICU admission

	No T before VAP		T before VAP	p-value
	No T after VAP	T after VAP		
Number of patients	36	62	15	
Men	20 (56)	44 (71)	5 (53)	0.207
Age (years)	72 (60-78)	67 (48-76)	70 (58-80)	0.282
Smokers	7 (19)	14 (23)	2 (13)	0.717
SAPS II	31 (27-39)	41 (27-50)	41 (35-58)	0.024
APACHE II score	12 (9-16)	16 (12-20)	17 (12-20)	0.024
Comorbidity				
Diabetes mellitus	4 (11)	9 (15)	6 (40)	0.033
Malignant disease	7 (19)	8 (19)	0	0.174
COPD	6 (17)	7 (11)	3 (20)	0.598
Chronic cardiac disease	11 (31)	13 (21)	4 (27)	0.561
Kidney failure	4 (11)	2 (3)	2 (13)	0.204
Hypertension	19 (53)	25 (40)	8 (53)	0.408
Alcoholism	3 (8)	10 (16)	2 (13)	0.548
Main reason for ICU admission				
Medical*	0	3 (5)	0	0.234
Trauma without surgery	0	5 (8)	0	
Surgery	36 (100)	54 (87)	15 (100)	
Type of surgical patients				
General surgical†	25 (69)	19 (35)	4 (27)	0.002
Neurosurgical	11 (31)	35 (65)	11 (73)	

Results are presented as median (25^{th}-75^{th} interquartile range) or as number (%); VAP = ventilator-associated pneumonia; T = tracheotomy; ICU = intensive care unit; SAPS II = Simplified Acute Physiology Score II; APACHE II score = Acute Physiology and Chronic Health Evaluation II score; COPD = chronic obstructive pulmonary disease; *acute respiratory failure, sepsis, state after resuscitation; †neck, thorax, abdomen

Characteristics of VAP patients established at admission and during ICU stay

Table 1 shows the characteristics of VAP patients established at ICU admission. Patient groups did not differ significantly according to gender, age, smoking habit, and prevalence of the majority of comorbidities, with the exception of diabetes mellitus. Diabetes mellitus was most prevalent in the "T before VAP" group. SAPS II and APACHE II scores (taken during the first 24 hours following ICU admission) were lower in the "no T after VAP" as compared to the "T after VAP" and the "T before VAP" groups. Reasons for ICU admission did not differ significantly between the groups (Table 1). During study period there were 105 (93%) surgical patients: 48 (46%) general surgical (including surgery of neck, thorax and abdomen) and 57 (54%) neurosurgical (including surgery of head) patients. The type of surgical patients significantly differed between the groups: surgery of neck, thorax and abdomen as the grounds for ICU admission prevailed in not tracheotomized patients (25/36, 69%), while neurosurgery-related ICU admissions were most often seen in tracheotomized patients (46/77, 60%) (p=0.002).

Table 2 shows the characteristics of VAP patients observed during the ICU stay. Reasons for MV and performing tracheotomy are shown in Table 2. Duration of MV before VAP and the number of days at ICU before VAP were the longest in the "T before VAP" group. The groups did not differ significantly in

Table 2. Characteristics of VAP patients according to tracheotomy (T) status during ICU stay

	No T before VAP		T before VAP	p-value
	No T after VAP	T after VAP		
Number of patients	36	62	15	
Reason for MV				
Respiratory*	26 (72)	35 (56)	8 (53)	0.163
Neurological†	6 (17)	12 (20)	1 (7)	
Sedation	4 (11)	15 (24)	6 (40)	
Number of days of MV before VAP	7 (3-11)	3 (2-5)	9 (6-14)	<0.001
Number of days at ICU before VAP	5 (4-7)	5 (4-9)	12 (10-24)	<0.001
Number of reintubations	2 (0-3)	1 (0-2)	0 (0-2)	0.098
Reason for T				
Respiratory‡	-	31 (50)	6 (40)	0.487
Neurologic§	-	31 (50)	9 (60)	
Number of days of MV before T	-	6 (4-9)	7 (4-7)	0.777
Number of days at ICU before T	-	9 (7-14)	8 (6-11)	0.242
Number of days from T to VAP	-	-	6 (3-13)	
Number of patients treated with antibiotics	36 (100)	62 (100)	15 (100)	1.000
Number of antibiotics before VAP	2 (0-3)	1 (0-2)	3 (2-5)	0.012
Number of antibiotics before T	-	2 (1-3)	2 (1-3)	0.919
Total number of antibiotics	3 (2-5)	3 (2-5)	4 (2-8)	0.738
Number of patients treated with corticosteroids	8 (22)	17 (27)	2 (13)	0.473
Number of sedated patients	7 (19)	26 (42)	9 (60)	0.012

Results are presented as median (25^{th}-75^{th} interquartile range, or as number (%); VAP = ventilator-associated pneumonia; ICU = intensive care unit; T = tracheotomy, MV = mechanical ventilation; *acute respiratory failure, neuromuscular weakness, acute respiratory distress syndrome; †Glasgow Coma Score (GCS) lower than 9; ‡predicted long MV due to trauma or disease of the lung (>10 days), weaning difficulties, neuromuscular weakness/need for frequent suctioning (>10-12 times daily); §GCS lower than 9

the reason for MV or tracheotomy, number of reintubations, number of days of MV or number of ICU days prior to tracheotomy, representation of corticosteroid-treated and antibiotic-treated patients. Patient groups did not differ significantly in total number of antibiotics and number of antibiotics before tracheotomy, but there was a higher number of antibiotics before VAP in "T before VAP" group as compared to the "no T before VAP" group. Also, during study period there was more sedation in "T before VAP" group.

Characteristics of VAP

Significant differences in CPIS scores between the groups failed to be found. The representation of all parameters of CPIS did not significantly differ between the groups (Table 3). In all patients with valid ETA (<10 squamous epithelial cells *per* visual field), the infection was polymicrobial. Eleven patients were excluded from final analysis of the etiology of VAP because they had growth of bacteria below the concentration of 10^5 CFU/mL. So, final analysis of bacterial etiology of VAP included 93 patients/ETA samples with 147 isolated bacterial species. In all patient groups, the most common causative agents were gram-negative bacteria (Table 4). *Pseudomonas aeruginosa* was the most prevalent gram-negative bacterium in all groups. Other frequently isolated gram-negative bacteria were *Acinetobacter* species, *Enterobacter* species and *Escherichia coli* in the "no T after VAP" group, *Escherichia coli*, *Haemophilus influenzae*, *Acinetobacter* species and *Escherichia coli* in the "T after VAP" group, and *Acinetobacter* species, *Escherichia coli* and *Haemophilus influenzae* in the "T before VAP" group. Among gram-positive bacteria, the most prevalent species seen across the study groups was *Staphylococcus aureus* (in up to 38% of analyzed samples). Methicillin-resistant *Staphylococcus aureus* (MRSA) was isolated in 15 of 31 (48.4%) cases of *Staphylococcus aureus* infection. MRSA was isolated in 6 out of 30 (20%) ETA samples in the "no T after VAP" group, 6 out

Table 3. Characteristics of VAP according to tracheotomy (T) status

	No T before VAP		T before VAP	p-value
	No T after VAP	T after VAP		
Number of patients	36	62	15	
CPIS score	7 (7-9)	8 (7-9)	8 (7-9)	
Temperature (°C)	37.5 (37.0-38.7)	38.0 (37.2-38.6)	38.5 (37.2-39.3)	0.425
Leukocyte number (*per* mm^3)	12 (9-16)	14 (10-19)	12 (10-18)	0.451
Tracheal secretion				
Rare	3 (8)	3 (5)	0	0.552
Abundant	8 (22)	12 (19)	5 (33)	
Abundant + purulent*	25 (69)	47 (76)	10 (67)	
PaO2/fiO2 (mm Hg)	209 (168-256)	195 (157-298)	170 (114-217)	0.238
Chest radiograph				
No infiltrate	2 (6)	4 (6)	1 (8)	0.345
Diffuse infiltrates	10 (28)	11 (18)	6 (40)	
Localized infiltrate	24 (67)	47 (76)	8 (53)	
ETA				
Number of valid ETA†	33 (92)	56 (93)	15 (100)	0.529
Number of valid positive ETA‡	30 (83)	50 (81)	13 (87)	0.979
Polymicrobial ETA	33 (100)	56 (100)	15 (100)	0.163

Results are presented as median (25^{th}-75^{th} interquartile range) or as number (%); VAP = ventilator-associated pneumonia; T = tracheotomy, ETA = endotracheobronchial aspirate; CPIS = Clinical Pulmonary Infection Score; *contains more than 25 neutrophils *per* visual field; †ETA with <10 squamous epithelial cells *per* visual field; ‡ETA with <10 squamous epithelial cells *per* visual field and pathogen isolated at a concentration of more than 105 CFU/mL

Table 4. Bacterial species isolated from ETA in VAP patients

	No T before VAP		T before VAP	p-value
	No T after VAP	T after VAP		
Number of ETA samples	30	50	13	
Total number of bacteria	50	76	21	
Gram-negative bacteria	**42 (84)**	**53 (70)**	**15 (71)**	
Moraxella catarrhalis	0	2 (4)	0	0.415
Haemophilus influenzae	1 (3)	10 (20)	2 (15)	0.113
Pseudomonas aeruginosa	14 (47)	10 (20)	4 (31)	0.042
Acinetobacter species	8 (27)	8 (16)	4 (31)	0.362
Stenotrophomonas malthophilia	2 (7)	0	0	0.117
Escherichia coli	5 (17)	8 (16)	3 (23)	0.830
Klebsiella species	4 (13)	5 (10)	0	0.395
Enterobacter species	6 (20)	5 (10)	1 (8)	0.362
Proteus mirabilis	0	2 (4)	0	0.415
Serratia species	2 (7)	1 (2)	0	0.404
Citrobacter species	0	2 (4)	0	0.415
Unspecified gram-negative bacteria	0	0	1 (8)	0.044
Gram-positive bacteria	**8 (16)**	**23 (30)**	**6 (29)**	
Staphylococcus aureus	7 (23)	19 (38)	5 (38)	0.369
Streptococcus pneumoniae	1 (3)	3 (6)	0	0.605
β-hemolytic *Streptococcus* group B	0	1 (2)	0	0.647
Unspecified gram-positive bacteria	0	0	1 (8)	0.044

Results are presented as the number of bacterial species isolates (% out of the number of ETA samples); VAP = ventilator-associated pneumonia; T = tracheotomy; ETA = endotracheobronchial aspirate

of 50 (12%) ETA samples in the "T after VAP" group and 3 out of 13 (23%) ETA samples in the "T before VAP" group (p=0.490). Among all bacteria, the most prevalent bacterium was *Pseudomonas aeruginosa* in the "no T after VAP" group (47% *vs*. 20% *vs*. 31%; p=0.042), while in the other two groups, the most prevalent bacterium was *Staphylococcus aureus* (found in 38% of analyzed ETA samples), but the difference did not reach statistical significance (p=0.369).

Outcomes of study patients

In VAP patients not tracheotomized during ICU stay, mortality rates were approximately two-times higher as compared with patients tracheotomized either before or after VAP onset (crude risk ratio 1.83, 95% confidence interval (95% CI) 1.15-2.91, p=0.01; crude odds ratio 3.47, 95% CI 1.52-7.94; p=0.003) (Table 5). This result was confirmed by multiple linear logistic regression analysis adjusted for tracheotomy, comorbidities (malignant disease, chronic cardiac disease), duration of MV and use of corticosteroids (P_{model}<0.001, Pseudo R^2=0.193). The model revealed the odds ratio for lethal outcome to be 0.19 (95% CI 0.07-0.51, p=0.001) in tracheotomized *versus* non-tracheotomized VAP patients (regardless of tracheotomy timing). Other significant predictors, all of them correlated to lethal outcomes, were duration of MV (OR 1.00, 95% CI 1.00-1.1, p=0.029) and use of corticosteroids (OR 0.3, 95% CI 0.1-0.8, p=0.019). In the surviving VAP patients, the length of ICU stay before VAP was twice shorter in the "no T before VAP" group as compared with the "T before VAP" patients. The number of days from VAP to ICU release was higher in the "T after VAP" group than in the non-tracheotomized and "T before VAP" patients. The number of pre-VAP days on MV was twice higher in

Table 5. Outcome of study patients

	No T before VAP		T before VAP	p-value
	No T after VAP	T after VAP		
Mortality	23 (64)	21 (34)	5 (33)	
Surviving VAP patients	13 (36)	41 (66)	10 (67)	0.011
Length of ICU stay (days)	15 (11-20)	21 (14-36)	22 (16-28)	0.113
Number of days at ICU before VAP	5 (3-7)	5 (4-11)	11 (10-13)	<0.001
Number of days at ICU before T	-	10 (6-13)	8 (5-10)	0.152
Number of days from VAP to ICU release	10 (6-15)	14 (10-27)	9 (3-13)	0.033
Number of days from T to ICU release	-	11 (6-16)	15 (9-19)	0.354
Total duration of MV (days)	7 (5-11)	9 (6-18)	8 (6-12)	0.747
Number of days of MV before VAP	3 (1-6)	4 (2-6)	8 (6-10)	0.001
Number of days of MV before T	-	6 (4-9)	6 (4-7)	0.566
Number of days of MV after VAP	4 (3-7)	4 (3-12)	0 (0-1)	0.001
Number of days of MV after T	-	2 (0-9)	2 (1-8)	0.853
SAPS II at release	21 (15-25)	26 (20-31)	23 (15-25)	0.231

Results are presented as median (25^{th}-75^{th} interquartile range) or as number (%); ICU = intensive care unit; VAP = ventilator-associated pneumonia; MV = mechanical ventilation; T = tracheotomy; SAPS II = Simplified Acute Physiology Score II

the patients tracheotomized before VAP onset than in the "no T before VAP" patients. The number of post-VAP days on MV was higher in the "no T before VAP" group as compared with the "T before VAP" group. In the "T before VAP" group, the number of post-VAP days on MV was 0 (0-1). Other endpoints analyzed in surviving patients, i.e. total length of ICU stay, number of ICU days before tracheotomy, number of days elapsed from tracheotomy to ICU release, total duration of MV, number of pre- and post-T days on MV and SAPS II at release, did not differ between the groups.

Discussion

Ventilator-associated pneumonia mortality rates range from 24% to 50%, and can reach 76% in certain specific settings or when VAP is caused by high-risk pathogens[1]. Some studies suggest the mortality to be increased in tracheotomized patients[25]. According to our data, in VAP patients not tracheotomized during ICU stay, the mortality was approximately two-fold higher than in patients tracheotomized either before or after VAP onset. Other predictors positively correlated with lethal outcomes were duration of MV and use of corticosteroids, which is consistent with the results of many other studies[3,13-15,29]. Koleff *et al.* also found the mortality of patients receiving tracheotomy to be lower than the mortality of non-tracheotomized patients[17]. One large retrospective observational cohort study found tracheotomy to be associated with an improved in-hospital survival[30].

Data on the surviving patients showed that the total duration of MV and total length of ICU stay were the same in all groups. The duration of MV before VAP, as well as the length of ICU stay before VAP, were two times longer in the "T before VAP" group as compared with the "no T before VAP" group, but the mortality was the same as in the " T after VAP" group or lower than in the "no T after VAP" group. It is also interesting to note that the duration of MV after VAP was shortest in the "T after VAP" group, suggesting that tracheotomy shortens the duration of MV after VAP and consequently yields better patient outcomes, but only if the procedure is performed prior to VAP onset. The ICU length of stay after VAP was longest in the "T after VAP" group, but probably only because tracheotomy in that group was done later than in the "T before VAP" group.

During the study period, the incidence of VAP in our ICU was 25%. According to current literature, the incidence of VAP generally ranges from 8% to 28%[1], but can be much higher, especially in surgical ICUs[3].

Therefore, the high rate of VAP found in our surgical ICU is in line with these data. In our ICU patients, tracheotomy reduced the relative risk of VAP onset by 67% (relative risk 0.33, 95% CI 0.20-0.56). Namely, in 453 ICU patients mechanically ventilated for >48 h, VAP developed in 98 of 290 (34%) "non-T" patients and in only 15 of 163 (9%) "T" patients. The above-stated relative risk of VAP onset, seen in our "T" and "non-T" ICU patients, represents a crude risk ratio, since data on the potential confounders in mechanically ventilated patients who did not develop VAP during ICU stay were not collected at this point.

According to the literature, the incidence of VAP among tracheotomized patients varies from 6% to 26%[9,31,32]. However, many studies failed to specify the time elapsed between tracheotomy and VAP onset**.** Only a small number of studies explicitly state whether tracheotomy preceded VAP or followed it[8,13,15,22,25,30]. To that matter, the work of Nseir *et al*. even warns that the incidence of VAP after tracheotomy never got to be compared with the incidence before tracheotomy or the incidence in patients without tracheotomy[8]. Pawar *et al*. found that most VAP cases (88.9%) occurred before tracheotomy, so that tracheotomy came as a result of VAP rather than posing a risk factor favoring its development[15]. According to our data, the incidence of VAP among tracheotomized patients was 68% (77/113), while the incidence of VAP among non-tracheotomized patients was 32% (36/113), the time elapsed between tracheotomy and VAP onset hereby not being taken into consideration. But, if we take into account only the patients that developed VAP after T, the incidence of VAP among tracheotomized patients would drop to 13% (15/113) only.

Lower SAPS II and APACHE II scores probably were the reasons why tracheotomy was not performed in the "no T after VAP" group. Despite lower SAPS II and APACHE II scores, the mortality among the non-tracheotomized patients was approximately two-times higher as compared with the patients tracheotomized either before or after VAP onset. Our data suggest that tracheotomy reduces mortality of VAP patients, even if performed following VAP onset.

The main reason for ICU admission differed between the groups; the most prevalent reason in tracheotomized patients was neurosurgery, probably because prolonged mechanical ventilation was to be expected. In non-tracheotomized VAP patients, on the other hand, the most prevalent ground for ICU admission was general surgery, probably because of respiratory problems not seen immediately after surgery, but in the later ICU stay course.

A higher use of sedatives was recorded in tracheotomized patients, especially in the "T before VAP" group; namely, since most of these patients were admitted to ICU due to neurotrauma, the severity of their injuries mandated intentional few-day sedation.

Approximately 58% of VAP-causing microorganisms isolated within the frame of various studies were gram-negative bacteria, among which *Pseudomonas aeruginosa* turned out to be the most frequent one, and the most frequently isolated gram-positive bacterium was MRSA[1,20,29]. VAP etiology uncovered within the frame of our study was pretty much the same. Also, in our study *Staphylococcus aureus* was the most frequently isolated species in tracheotomized patients, accounting for 38% (24/63) of ETA isolates. According to Park *et al*., one of the risk factors for VAP caused by *Staphylococcus aureus* is neurosurgical procedure as a reason for ICU admission, and our study group of tracheotomized patients included 66% (51/77) of neurosurgical patients[33].

Conclusion

Although many studies have shown tracheotomy to be a major risk factor for VAP development[13-19], our study results do not support that claim. Our data suggest that lower VAP incidences are to be expected in tracheotomized patients. According to our data, tracheotomized VAP patients have reduced mortality rates regardless of the tracheotomy being performed before or after VAP onset. Also, the present study indicated tracheotomy to be associated with a reduced duration of mechanical ventilation after VAP onset, but only if patients were tracheotomized at the moment of VAP onset.

Our study had some limitations in terms of its retrospective and observational design, the heterogeneity of reasons for ICU admission (general surgical and neurosurgical grounds), a single ICU coverage, and a small sample size, especially when it comes to the "T before VAP" group.

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Sažetak

UTJECAJ TRAHEOTOMIJE NA KLINIČKI TIJEK VENTILACIJSKE PNEUMONIJE

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Ventilacijska pneumonija (*ventilator-associated pneumonia*, VAP) je jedna od najčešćih infekcija među bolesnicima u jedinicama intenzivnog liječenja (JIL). Cilj ovoga istraživanja je bio utvrditi utjecaj traheotomije na klinički tijek VAP-a. Istraživanje je provedeno u 15-krevetnoj Jedinici intenzivnog liječenja Odjela za anesteziologiju, reanimatologiju i intenzivno liječenje u Kliničkom bolničkom centru "Sestre milosrdnice", Zagreb, Hrvatska. Svi bolesnici u kojih se razvila VAP tijekom navedenog razdoblja bili su uključeni u istraživanje. U bolesnika s VAP koji nisu traheotomirani (T) tijekom njihovog boravka u JIL-u smrtnost je bila otprilike dva puta veća u usporedbi s bolesnicima koji su traheotomirani prije ili nakon razvoja VAP (*crude risk ratio* 1,83, 95% *confidence interval* (CI) 1,15-2,91, p=0,01; *crude odds ratio* 3,47, 95% CI 1,52-7,94; p=0,003). Među preživjelim bolesnicima trajanje mehaničke ventilacije prije razvoja VAP je bilo duže u skupini "T prije VAP" u usporedbi sa skupinom "bez T prije VAP" (8, 6-10 prema 3, 2-5; p<0,001), ali je broj dana mehaničke ventilacije nakon razvoja VAP bio kraći u bolesnika skupine "T prije VAP" u usporedbi s onima skupine "bez T prije VAP" (0, 0-1 prema 4, 3-9; p<0,001). Trajanje mehaničke ventilacije nakon razvoja VAP u skupini "T nakon VAP" je bilo duže u usporedbi sa skupinom "T prije VAP" (4, 3-12 prema 0, 0-1; p<0,001). Ovo istraživanje je ukazalo na to da je traheotomija povezana s kraćim trajanjem mehaničke ventilacije nakon pojave VAP, ali samo ako su bolesnici u trenutku pojave VAP traheotomirani.

Ključne riječi: *Intenzivna skrb; Traheotomija; Pneumonija, ventilacijska; Prognoza; Preživljavanje, analiza; Ishod liječenja*